Endovascular Treatment of AVMs in Glasgow

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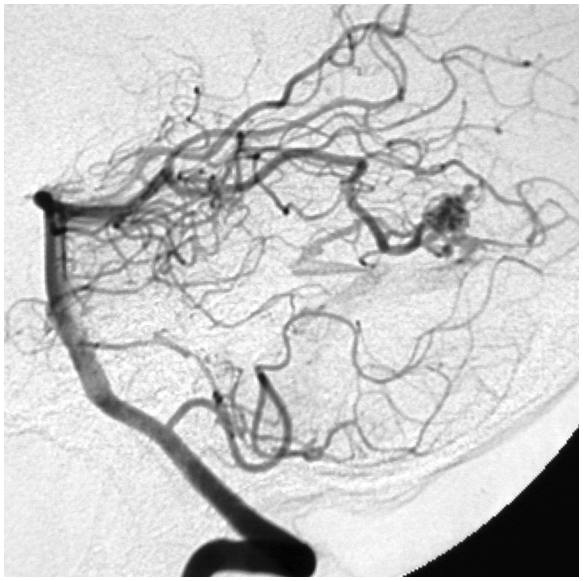
Introduction

The Institute of Neurological Sciences (INS) at the Southern General Hospital in Glasgow is the largest Neuroscience Centre in Scotland, and among the largest in the UK. The other 3 Scottish centres are Aberdeen, Dundee and Edinburgh. Scotland itself has a population of approximately 5.2 million people and the INS in Glasgow provides neurointervention cover to almost 3 million of these. Referrals also come from the rest of Britain and overseas (North Africa, Middle-East and Asia). Glasgow has been nominated as one of two centres in the UK for management of neonatal high-flow fistulae, and has the largest experience in the UK for management of cervico-facial vascular malformations.

There are 3 consultant interventional neuroradiologists (in addition to 6 purely diagnostic neuroradiologists) and two fellows (at least one of whom is training in neurointervention). Overseas trainees are sometimes accommodated, most recently from Greece and Thailand. Patients requiring interventional neuroradiology are usually admitted into the neurosurgery unit, although care is shared with the neuroradiologists. An AVM clinic is run jointly with neurosurgical colleagues, to which most (but not all) AVM patients are referred, and close links are maintained with the National Stereotactic Radiosurgery Centre in Sheffield (which has overwhelmingly, the largest experience in the UK for the radiosurgical treatment of AVMs). The Scottish Intracranial Vascular Malformations Study (SIVMS) is a collaborative epidemiological study between the four Scottish centres[1,2,5].

Procedure overview in Glasgow

Patients are normally admitted the day before AVM embolization for consent and anaesthetic review. We perform all procedures under general anaesthesia and provocative (Amytal) testing is never performed. All our AVM procedures are performed with systemic heparinisation (ACT 2× baseline) which is reversed at the end of the procedure, and with steroid (Dexamethasone) cover (which continues for about 3 days). A 6F guidecatheter (Cordis Envoy or Boston Guider Soft-tip) is placed in the parent vessel. The microcatheters we use are mainly Spinnaker 1.5 or 1.8 (Boston Scientific) with Mizzen 12 soft wire (Boston Scientific) or Magic 1.2fm (Balt) with Mirage 008 wire (MTI). Baltacci (Balt), Ultraflow and other wire-guided catheters are less commonly used. We perform AVM embolization with NBCA glue. Where possible we use Glubran-2 (which is both effective, and has CE-marking), but with high-flow fistulae or other uncertain situations we use Histoacryl (for its long established reliability). Our glue concentrations (diluted with Lipiodol) range from 20% to 100% (pure glue being opacified with Tantalum or more recently Tungsten), while injection durations range from rapid



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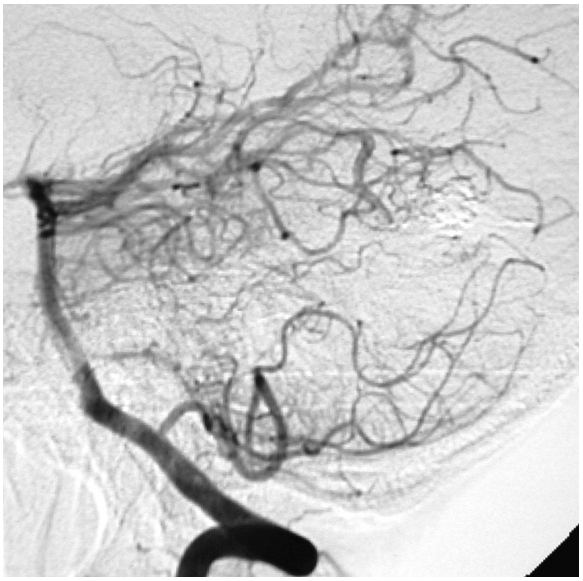

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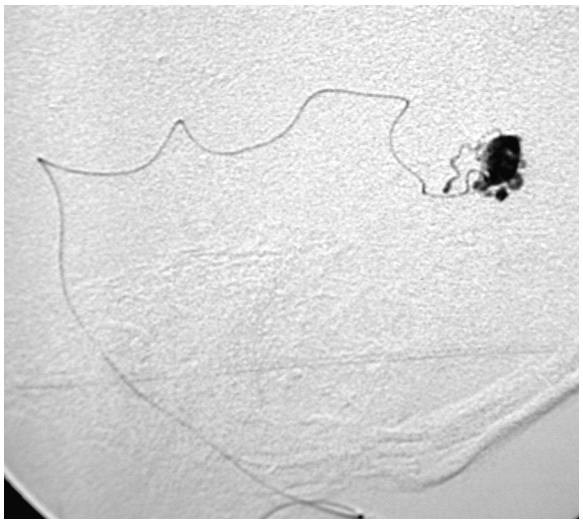

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Figure 1 27-year-old woman who presented with seizures. MRI revealed a small occipital AVM related to the tentorium cerebelli. A) Pre-embolization angiogram shows single feeder. B) Microcatheter (Magic 1.2) navigated with Mirage wire into nidus. A single injection of Glubran-2 sealed the AVM. C) Follow-up angiography at 1 year confirms cure.

short injections for high-flow fistulae to 2 minutes or more for a true nidus. The microcatheter only, is withdrawn following a glue injection, the guide catheter remaining in situ for the next microcatheter. Normally, no more than four glue injections are performed at one session with further sessions scheduled at 2 or 3 month intervals. Following the embolisation procedure, the patient is extubated, but blood pressure is controlled with sedation and Nimodipine infusion

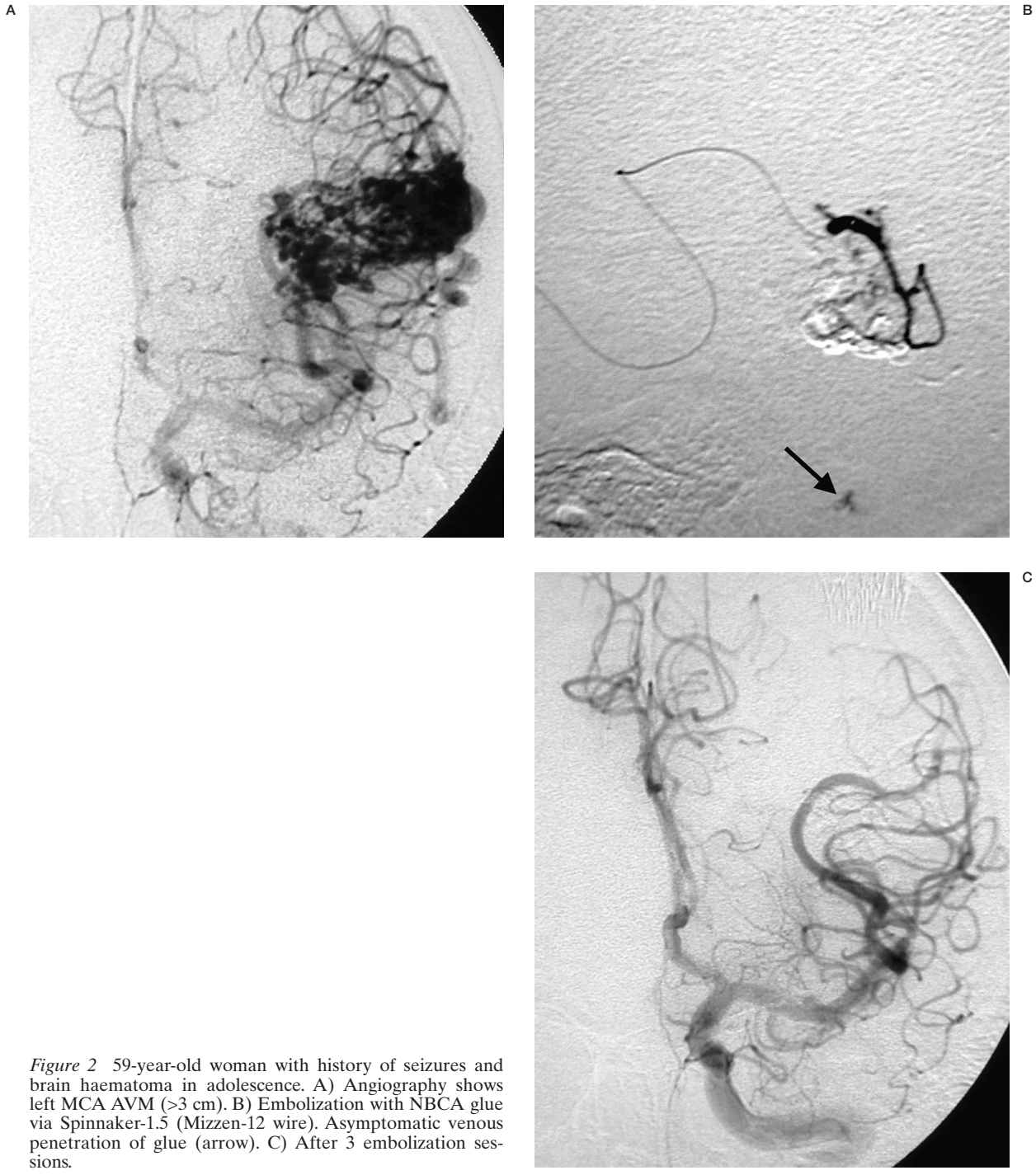

Figure 2 59-year-old woman with history of seizures and brain haematoma in adolescence. A) Angiography shows left MCA AVM (>3 cm). B) Embolization with NBCA glue via Spinnaker-1.5 (Mizzen-12 wire). Asymptomatic venous penetration of glue (arrow). C) After 3 embolization sessions.

as necessary in the High Dependency ward (adjacent to ITU).

After 24 hours the patient returns to the Neurosurgery ward and is normally discharged one or two days later.

Number of Cases

Over the last 7 years approximately 250 patients with brain AVMs have been referred or admitted. Of these, 127 patients have under-

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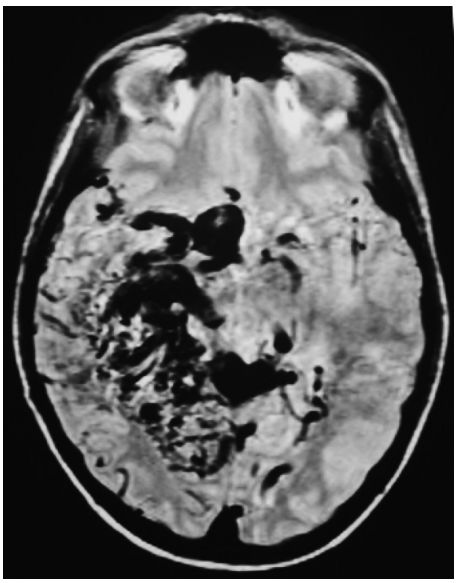

Figure 3 A 42-year-old woman with severe seizures poorly controlled (4-5 seizures per week) on maximal drug therapy. A) MRI shows large incurable AVM. B) Vertebral angiogram shows large occipito-temporal deep AVM and giant basilar aneurysm. C) After 3 embolization sessions nidal flow markedly reduced. Seizure frequency reduced to one in three months.

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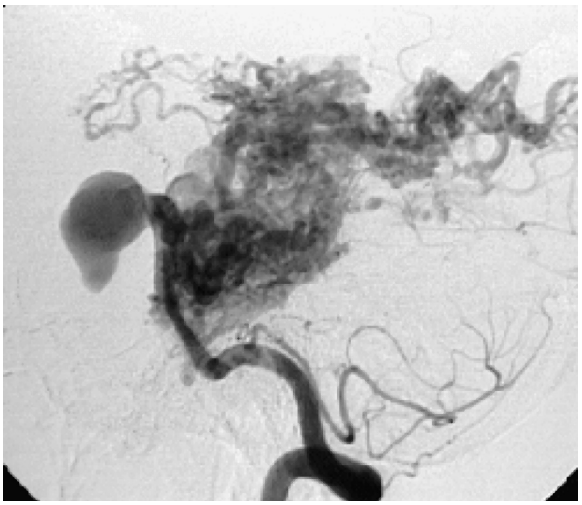

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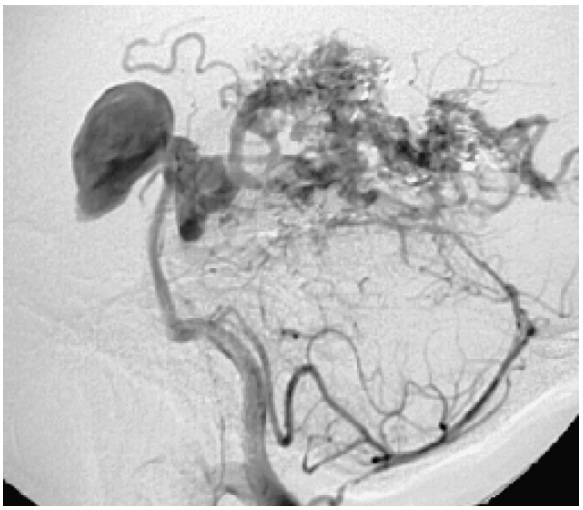


gone embolization treatment. The others had surgery, radiosurgery or conservative treatment. Overall 207 separate embolisation procedures were performed ie. 1.6 procedures per patient. Fewer patients were treated in the first few years and the workload has stabilized at around 40-50 procedures per year. The number of glue injections/microcatheters per procedure varied from 1 to 6 (mean 2.5) with 530 individual microcatheter glue injections performed in total. 23 procedures have also been performed for neonatal or infantile high flow fistulae (vein of Galen malformations).

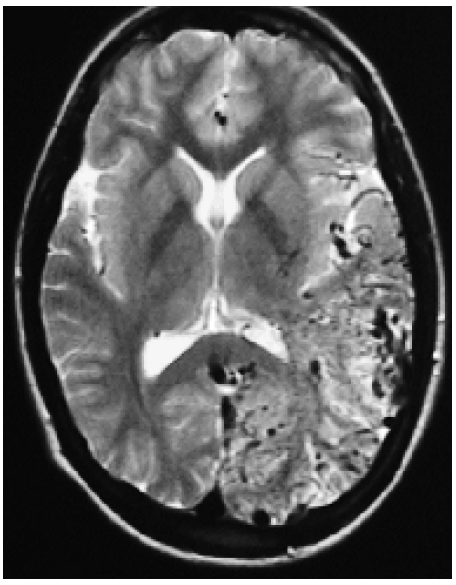
Age Range and Mean Age

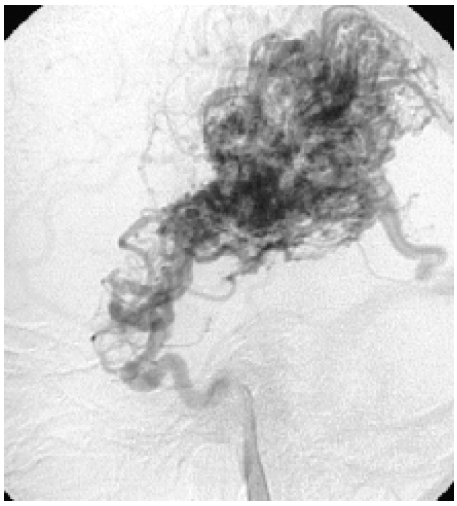
Overall age range is 3 days to 70 years, which includes neonates with Vein of Galen Malformations. Excluding neonatal and infantile patients, mean age at presentation was 43 years.

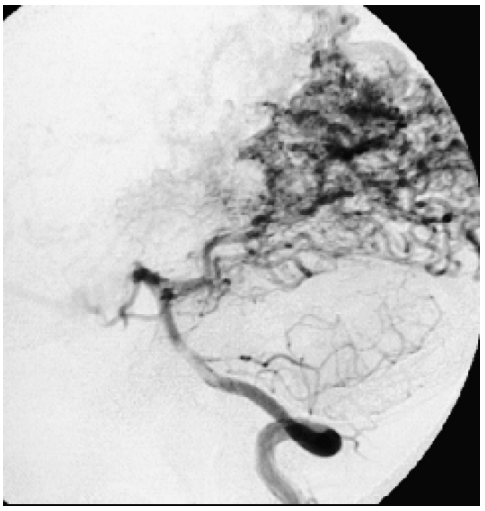
Clinical Presentation

Haemorrhage	45.5%
Epilepsy	37%
focal neurological deficits	8.5%
headaches	5%

Figure 4 A 24-year-old woman with severe migrainous headaches and a proliferative angiopathy pattern AVM. A) T2-weighted MRI demonstrates AVM nidus interspersed with brain parenchyma. Functional MRI confirmed functioning brain tissue within nidus. B, C) Angiography demonstrates large diffuse nidus.







other symptoms (e.g. tinnitus)	2%
asymptomatic	2%

Follow-up period

After complete morphological cure, follow-up angiography is performed in adult patients at one year post embolization; patients are then reviewed in the AVM clinic prior to final discharge. Children and adolescents when cured have follow-up angiography at one and five years post treatment and we recommend further angiographic assessment when they reach adulthood. This is important as an AVM in childhood, may not show complete morphological expression: recurrent AVMs following apparently complete cure are well recognized in children.

Results

For small AVMs (<3 cm diameter) complete occlusion is achieved in 62% cases (figure 1). Micro AVMs (<1 cm diameter) are rare in our series but are usually completely sealed in a single session.

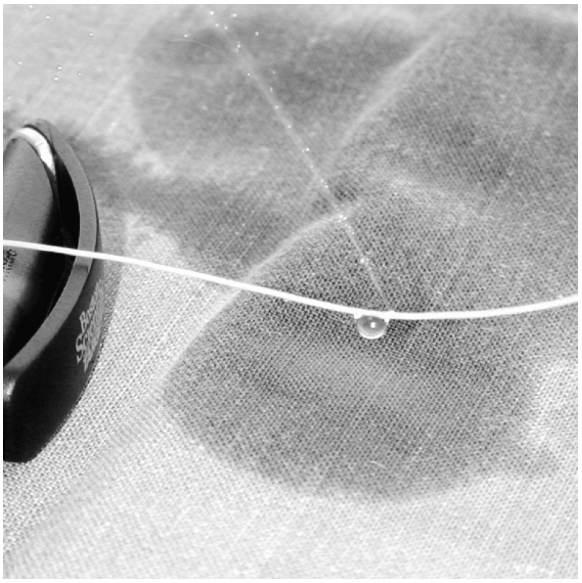


Figure 5 A Spinnaker-1.8 microcatheter being flushed with saline solution prior to use. The proximal leak is readily visualised.

For AVMs larger than 3cm, embolisation is usually part of a multimodality treatment with surgery or radiosurgery. Embolization alone is curative in only about 15% (figure 2). Progression to complete occlusion after partial embolisation was seen in 2 cases, one of which had been referred for radiosurgery.

Embolic Material(s) used

Cyanoacrylate glue (Glubran-2 or Histoacryl) opacified with Lipiodol (see above).

Therapeutic Strategy

Our overall therapeutic strategy varies according to the patient's age, presentation and the morphology of the AVM. Complete morphological cure is usually the preferred goal. For small AVMs (<3 cm) curative embolisation is the aim. For larger AVMs embolisation usually precedes surgery or radiosurgery. For cases presenting with haemorrhage, radiosurgery is not our preferred option unless the lesion is inaccessible to other treatment. We have seen rebleeds during the latent period following radiosurgery on several occasions. Where cure is not deemed possible we may offer partial targeted treatment for nidal aneurysms or AV fistulae. On several occasions we have treated large uncurable AVMs in an attempt to control intractable seizures (figure 3).

We normally avoid treatment for large diffuse AVMs. Identification of patients with proliferative angiopathy is important we believe, as is the recognition of perinidal angiogenesis. This is not mere dogma, and ignorance of these morphological patterns exposes patients to unnecessary risks. In our series of 250 brain AVMs we identified 8 patients with proliferative angiopathy (large diffuse nidus, interspersed normal brain tissue, arterial occlusive disease and sprouting angiogenesis) (figure 4).

These patients had a distinct course presenting earlier (mean age: 25 years), and rarely with haemorrhage (*abstract accepted for WFITN Congress 2006*). We have usually avoided treatment in these cases as well as in diffuse deep AVMs as seen in CAMS (Wyburn-Mason syndrome)[8].

Technical Complications during Embolization Procedures

On two occasions, aneurysms ruptured during AVM embolisation. In one case, after an uneventful glue embolization, during final control angiograms anaesthesia became prematurely lightened. patient began coughing elevating blood pressure and an incidental PCom aneurysm ruptured. Heparin was reversed immediately and the aneurysm coiled. Apart from headache, the patient was asymptomatic and made a complete and uneventful recovery. In the other case, the patient had presented with subarachnoid haemorrhage WFNS grade III related to a cerebellar AVM and treatment was performed during the acute phase. There were co-existing flow aneurysms on the feeding PICA. During embolization of the nidus, one of the aneurysms ruptured. The patients condition declined progressively over the next few days and she died on day 4. No vessel perforations or nidal ruptures were seen during embolisation. In the 530 microcatheter glue injections, 2 microcatheters (0.4%) were glued in place without clinical sequelae. No thromboembolic episodes were identified. Integrity of microcatheters is always checked carefully with saline flushes. One microcatheter (Spinnaker 1.8) was found to have a proximal leak (figure 5), re-emphasizing to us the importance of catheter checking.

Morbidity and Mortality

There was one death in 127 patients treated (0.8%), or 207 embolization procedures (0.5%), related to rupture of a co-existing aneurysm, as described above. There were 4 post-embolization haemorrhages over the next 24 hours (3.1% of patients, 1.9% of procedures). Three of these were symptomatic and one was discovered on MRI performed for other indication. In 2 cases, surgical evacuation of haematoma was required. 3 patients made a good recovery (GOS5) 1 made a moderate recovery (GOS4). 7 other procedures (5.5% of patients, 3.4% of procedures) developed new or worsening neurological deficits post embolization, with no evidence of haemorrhage. 5 of these made a good recovery (GOS5) and two a GOS4. In 2 of these 7 the deficit was associated with perinidal oedema which resolved together with the deficit, with prolonged steroid treatment.

References

1 Al-Shahi R, Bhattacharya JJ et Al: Prospective, population-based detection of intracranial vascular malformations in adults: The Scottish Intracranial Vascular Malformation Study (SIVMS). Stroke 34: 1163-1169, 2003.
2 Al-Shahi R, Bhattacharya JJ et Al: Scottish Intracranial Vascular Malformation Study (SIVMS): Evaluation of methods, ICD-10 coding, and potential sources of bias in a prospective, population-based cohort. Stroke 34: 1156-1162, 2003.
3 Bhattacharya JJ, Thammaroj J: Vein of Galen malformations. J Neurol Neurosurg Psych 74: 42-44, 2003.
4 Thammaroj J, Bhattacharya JJ: Interventional neuroradiology in neonates and children. Radiology Now 19: 8-11, 2002.
5 Al-Shahi R, Pal N et Al: Observer agreement in the angiographic assessment of arteriovenous malformations of the brain. Stroke 33, 1501-1508, 2002.
6 Stone J, Bhattacharya J et Al: Syndrome in a female: Relationship to Sneddon's syndrome and radiographic appearances. Neuroradiol 43: 562-564, 2001.
7 Chul Suh D, Alvarez H et Al: Intracranial haemorrhage within the first two years of life. Acta Neurochirurg 143: 997-1004, 2001.
8 Bhattacharya JJ, Luo CB et Al: Wyburn-Mason or Bonnet-Dechaume-Blanc as Cerebrofacial Arteriovenous Metameric Syndromes (CAMS): A new concept and a new classification. Intervent Neuroradiol 7: 5-17, 2001.
9 Bhattacharya JJ, Lamin S et Al: Cyanoacrylate glue in the embolisation of small brain AVMs. Presented at WFITN 2003 Recife, Brazil.
10 Zampakis P, Bhattacharya JJ et Al: Proliferative angiopathy: a single centre experience of diffuse brain AVMs (abstract accepted for WFITN Congress 2006).
11 Stenhouse E, Bhattacharya JJ et Al: Antenatal MRI in the management of vein of Galen malformations (abstract accepted for BSNR Congress 2006).

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